cycling. Also, while gabapentin may be safely used in combination with depakote and carbamazepine, their doses need adjustment when lamotrigine is initiated. Valproic acid should be reduced in dosage before starting lamotrigine to decrease inhibition of metabolism and reduce the risk of rash. Due to enzyme induction, the dose of carbamzepine may need to be reduced when lamotrigine treatment is initiated. The relatively recent use of gabapentin and lamotrigine for mood disorders limits the information regarding their long term therapeutic efficacy, and potential emergent side effects.

Other modalities being studied as mood stabilizers include calcium channel blockers, and thyrotropin releasing hormone (TRH) plus other endogenous neuropeptides. Calcium channel blockers like verapamil and nimodipine are showing special promise for rapid and ultra rapid cycling. Preliminary findings have suggested that the dihydropyridine class of L-type calcium channel blockers, which includes nimodipine, isradipine etc., compared to the phenylalkalamine verapamil may have greater mood stabilizing effects, and potential as an alternative or adjunct to lithium. Preliminary data on TRH indicate possible acute anti-depressant, anti-anxiety, and anti-suicide effects.

Although the search for safer alternatives to lithium therapy in affective disorders is increasingly promising, further research of the newer mood stabilizing medications is warranted in order to establish their safety and efficacy.

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Traumatic Disorders—Contemporary Directions

EMOTIONAL REACTIONS to trauma have been recorded since ancient times. In this century, the study of World War I veterans ("shell shock"), World War II consequences ("traumatic neurosis"), and later DSM-I (Gross Stress Reaction), DSM-II ("Transient Situational Disturbance"), Holocaust Survivors ("Survivors' Syndrome"), and DSM-III (PTSD) all constituted additions to a crystallized modern understanding of trauma reactions.

Since 1980, when Post Traumatic Stress Disorder (PTSD) became part of the official psychiatric nomenclature (DSM-III), a concerted effort has been made by specialists in psychiatric trauma to legitimize PTSD as a complex psychiatric disorder. Trauma can result in a range of disorders. The most commonly associated conditions with trauma are: PTSD, Acute Stress Disorder, other Anxiety Disorders, Mood Disorders, Somatization Disorders, Dissociative Disorders, Substance Abuse, and Adjustment Disorders. On the other hand, early childhood exposure to severe trauma more often results in affective psychopathology with little resemblance to PTSD. The type of traumatic psychopathology may be determined by the interaction of factors: type of trauma, age of impact, preexisting biological vulnerability, and possibly the complexity of affected brain mechanisms.

Diagnosis of Post Traumatic Stress Disorder represents the prototype of traumatic disorders. The symptomatology includes a classical triad of symptoms: a) The re-experiencing of the traumatic event, b) Both persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness, c) Symptoms of hyperarousal (difficulty falling asleep, insomnia, irritability, angry outbursts, etc.). These symptoms occur after the exposure via experiencing, witnessing, or confronting an event that is perceived as threatening and is accompanied by intense fear, horror, and helplessness. Prevalence of PTSD is 7.2 percent in the generation population. Between 50 and 90 percent of patients with PTSD have comorbid disorders such as anxiety, depression and alcohol abuse.

Other traumatic disorders include acute stress disorder, a condition that resembles PTSD but is characterized by a one-month duration (following the trauma) and includes significant dissociative symptoms. Anxiety disorders (more often panic disorders and generalized anxiety) are often the main psychiatric manifestation after traumatic stress. Most anxiety disorders occur more often in combination with other diagnoses rather than "pure culture" clinical forms. While, for example, major depression may not exhibit a different picture after a known traumatic stress, a history of traumatic stress may add additional biological abnormalities and may thus influence significantly the outcome of treatment.

After major traumatic events such as a catastrophe affecting an entire community, PTSD is relatively rare. Emotional manifestations of some kind are common, however. The majority of traumatized individuals will present a transient benign reaction characterized by terror, a sense of unreality, some depersonalization, some re-experiencing, transient anxiety, a sense of loss, and some preoccupation with images of the trauma that subsides within about 3 to 4 months. Risk factors for patients to PTSD after a community trauma include past psychiatric history, family psychiatric history, genetics, personality style such as neuroticism, introversion, personality disorder, history of trauma, low socioeconomic status, low intelligence, and even a family history of trauma.

PTSD, the prototype of traumatic disorders, is accompanied by biological markers such as an increase in blood pressure, pulse, glucocorticoid receptors, T3 and analgesia and reflect a marked sympathetic automatic overdrive, and a decrease in cortisol, alpha 2 adrenergic receptor activity, and stage three and four of sleep. In addition, acute stress is characterized by significant elevation of cortisol. Individuals suffering from chronic PSTD, on the other hand, have blunted plasma cortisol.

Recent and preliminary studies show that PTSD patients in veterans' hospitals have an average of 15 severe traumatic events in their lifetimes. Thus, in comparison to controls, individuals who develop PTSD are more likely to have had previous trauma. Finally, variants of PTSD occur. "Compassion fatigue," a form of traumatic disorder, may develop in health and mental health professionals who care for

patients with PTSD; "Disorder of extreme stress, not otherwise specified" (DESNOS or "Complex PTSD"), a condition that includes symptoms of PTSD associated with severe psychiatric complications, i.e., bipolar symptoms, psychosis, hallucinations, etc. also occurs. Secondary traumatization is another variant and refers to the occurrence of traumatic symptoms, more often PTSD, in individuals exposed to or who live with victims of traumatic stress.

In PTSD the process of memory consolidation may lead to continuous retraumatization, rendering PTSD a self-maintained condition similar to an autoimmune disorder. Early administration of beta blocking agents (limiting the adrenergic overdrive which is implicated in traumatic memory consolidation) and SSRIs (Fluoxetine, Sertraline, Paroxetine, Fluvoxamine), which work both on re-experiencing and avoidant sysmptoms, are now standard medical intervention. Long term use of minor tranquilizers is not indicated. Comorbid disorders have to be addressed at the same time (e.g., substance abuse). Most often clinicians are using a combination of dynamic and behavioral psychotherapy. Recalling the traumatic events in therapy or exposure to "reminders" has to be done carefully and gradually; therapists usually learn the amount of discomfort that such patients can tolerate. Other treatment modalities, e.g., EMDR (Eye Movement Desensitization Reprocessing) seem to have numerous enthusiasts but still remain to many authors controversial.

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When Chest Pain, Shortness of Breath, and Palpitations are Not Due to Cardiopulmonary Pathology

IT IS NOT UNCOMMON for the primary care physician, cardiologist, or emergency physician to see patients presenting with acute acute onset of chest pain, shortness of breath, palpitations, choking sensation, diaphoresis, trembling, paresthesias, lightheadedness, abdominal distress and fear of dying, only to find, after extensive and expensive workup, that there is no cardiopulmonary pathology present. Such patients often receive the diagnosis, "rule-out myocardial infarction," or "chest pain of unknown etiology." Panic disorder is characterized by recurrent and unexpected panic attacks (composed of at least four of thirteen symptoms, ten of which are listed above, and also include chills.

Panic disorder is defined by recurrent and unexpected panic attacks (composed of at least four of 13 symptoms, including chills or hot flashes, fear of losing control or going crazy, and de-realization or de-personalization) with at least one month of excessive concern about having another panic attack, or behavioral changes resulting from the attacks that are not substance-induced (e.g. precipitated by stimulants) and are not due to medical conditions (such as cardiac or vestibular disorders, hyperthyroidism, hyperparathyroidism, pheochromocytoma, or seizures.) Limited symptom attacks have fewer than four symptoms and are usually shorter and less severe. Panic attacks usually happen quickly (escalate in less than one minute and peak in less than 10 minutes) and typically last for 30 to 60 minutes, although some last for several hours. Nearly half of patients with panic disorder also have agoraphobia (fear of being in public places).

Panic disorder is common, with one-year prevalence at 1.5% of the population and lifetime prevalence at 2.5%. Women account for more than two-thirds of patients. Peak onset is from the late teens to mid-thirties, although it may come on at any age. There is a four to seven times greater incidence in first degree relatives.

The course of panic disorder is usually chronic, with waxing and waning, although one-third of patients go into complete remission. Co-morbid depression is common; two-thirds of panic disorder patients have concurrent major depressive disorder at some point in their illness, and one-third at any point in time. Depressed patients with panic disorder have a much higher suicide rate than those without panic disorder. Alcohol abuse occurs in one-third of panic disorder patients.

Various studies have pointed to α_2-adrenergic receptor dysfunction, postsynaptic serotonin receptor supersensitivity, blunted growth hormone response to Clonidine, and asymmetric blood flow and metabolism in the hippocampus (left lower than right) noted on PET scan. One study showed an elevation of neuropeptide Y in panic patients: neuropeptide Y is closely associated with noradrenergic activity in the central and peripheral nervous systems.

Hypochondriasis is common in panic patients. An average patient with panic disorder has 27 different unexplained symptoms. In various studies, 15% of patients in a vestibular disorder clinic and 20% of patients with dizziness had panic disorder, and panic patients were found to have a high incidence of gastrointestinal disturbance. Among patients referred for pulmonary function testing to a major medical center, 41% were found to have panic attacks, 11% were diagnosed with panic disorder, and 67% of patients with chronic obstructive pulmonary disease were found to have concurrent panic disorder. Of those patients seeing cardiologists for chest pain, 30–60% with negative workups have panic disorder; of those seeing gastroenterologists for irritable bowel syndrome 33–45% have panic disorder. While only 0.2% of those undergoing workup of pheochromocytoma actually have this condition, 67% have panic disorder.

Treatment of panic disorder has focused on antidepressants, benzodiazepines, and psychotherapy. Imipramine was found in several studies to have an excellent track record in the treatment of panic disorder with significant effects at moderate (1.5 mg/kg/D) to high (3 mg/kg/D) doses, with drop-out rates due to side effects 15% and 35% for moderate and high doses, respectively. Clomipramine at similar doses has also been shown to be very effective; other tricyclics and trazodone appear to be less effective. Monoamine oxidase inhibitors (MAOI), especially phenelzine, have shown similar efficacy. Due to the high side effect profile of tricyclics and MAOI's, and their high rate of fatality when taken in overdose, studies in the past few